far as their response to growth factors, plasma factors, and substrate are concerned.

22434. TSAI, SCHICKWANN*, STEPHEN G. EMERSON, COLIN A. SIEFF and DAVID G. NATHAN. (Div. Hematol. Oncol., Child. Hosp., Boston, Mass. 02115, USA.) J CELL PHYSIOL 127(1): 137–145. 1986. **Isolation of a human stromal cell strain secreting hemopoietic growth factors.**—A diploid fibroblastoid cell strain, termed "ST–1", has been established from a long–term liquid culture of human fetal liver cells. ST–1 cells are nonphagocytic, nonspecific esterase negative and do not possess factor VIII–related antigen but stain with antibodies specific for fibronectin and type I collagen. The ST–1 cells produce nondialyzable hemopoietic growth factors capable of stimulating the development of erythroid bursts, mixed granulocyte–macrophage colonies, pure granulocyte colonies, and pure macrophage colonies. These factors are active on both human fetal liver and human adult bone marrow progenitors. When liquid cultures of human fetal liver hemopoietic progenitors are established with a performed monolayer of ST–1 cells, the yields of nonadherent cells, erythroid progenitors, and myeloid progenitors are greatly increased. These studies demonstrate that the fibroblastoid ST–1 cells support hemopoiesis in vitro and may be a critical element in the stromal microenvironment in vivo.

22435. ZIPPEL, RENATA*, EMMAPAOLA STURANI, LUISELLA TOSCHI, LUIGI NALDINI, LILIA ALBERGHINA and PAOLO M. COMOGLIO. (Dip. Fisiol. Biochimica Generali, Sezione Biochimica Comparata, Univ. Milano, V. Celoria 26, 20133 Milano.) BIOCHIM BIOPHYS ACTA 881(1): 54–61. 1986. **In vivo phosphorylation and dephosphorylation of the platelet–derived growth factor receptor studied by immunoblot analysis with phosphotyrosine antibodies.**—Antibodies against the synthetic hapten azobenzyl phosphonate which specifically crossreact with phosphotyrosine have been produced and used to detect the proteins phosphorylated in tyrosine following exposure of intact quiescent Swiss 3T3 fibroblasts to the platelet–derived growth factor (PDGF). Western blotting of sodium dodecyl sulfate–polyacrylamide gel electrophoresis–fractionated proteins followed by decoration with phosphotyrosine antibodies and ^{125}I–labeled protein A have been used. The major tyrosine–phosphorylated component was a 170 kDa protein. The following lines of evidence suggest that this protein is the PDGF receptor in its tyrosine–phosphorylated form: (a) both proteins have the same (170 kDa) molecular weight; (b) the phosphorylated 170 kDa protein was detectable only in cell lines bearing the PDGF receptor; (c) the phosphorylation of the 170 kDa protein required PDGF and was dose–dependent. Kinetic studies showed that the phosphorylation of the receptor was maximal after 5–10 min at 37° C and was followed by a rapid decrement of the band. The loss of the 170 kDa component was not prevented by inhibitors of membrane internalization and of lysosomal proteinases, while it was inhibited by lowering the temperature to 5° C. In PDGF–stimulated cells, phosphotyrosine antibodies detected also a minor 36 kDa component phosphorylated at tyrosine.

22436. SOTO, EMILANO A., HARVEY J. KLIMAN, JEROME F. STRAUSS, III* and LAURIE G. PAAVOLA. (Dep. Obstet. Gynecol., Hosp. Univ. Pa., 3400 Spruce St., Philadelphia, Pa. 19104, USA.) BIOL REPROD 34(3): 559–570. 1986. **Gonadotropins and cyclic 3',5'–AMP alter the morphology of cultured human granulosa cells.**—Morphological changes in human granulosa cells in culture were observed by phase, fluorescent, scanning electron and transmission electron microscopy following the addition of human chorionic gonadotropin (hCG), luteinizing hormone (LH), 8–bromocyclic adenosine 3',5'–monophosphate (cAMP) and cytochalasins B and D. In response to these agents, polygon–shaped granulosa cells with granular cytoplasm became rounded, leaving fingerlike processes attached to the substratum and adjacent cells. The changes in cell shape were accompanied by a centripetal movement of mitochondria and lysosomes to a perinuclear location. The morphological alterations appeared to be mediated by cyclic AMP and to be the result of a dismantling and reorganization of microfilament–containing stress fibers. Follicle–stimulating hormone (FSH), prolactin (PRL), growth hormone (GH), and human placental lactogen (hPL) did not provoke cell shape changes. We conclude that tropic hormones capable of stimulating progestin secretion by luteinized granulosa cells cause a change in cell structure in vivo which leads to a redistribution of organelles involved in steroid synthesis. The possible relationship of the cytoskeleton to steroidogenesis is considered.

22437. KANEKO, SHIGERU, NOZOMI SATO, KATSUO SATO and INORU HASHIMOTO*. (Lab. Vet. Physiol., Kitasato Univ. Sch. Vet. Med. Anim. Sci., Towada–shi, Aomori 034, Jpn.) BIOL REPROD 34(3): 488–494. 1986. **Changes in plasma progesterone, estradiol, FSH and luteinizing hormone during diestrus and ovulation in rats with 5–day estrous cycles: Effect of antibody against progesterone.**—Progesterone secretion remained significantly higher during diestrus in the 5–day cyclic rat than in the 4–day cyclic animal. Injection of a sufficient amount of antiprogesterone serum (APS) at 2300 h on metestrus in a 5–day cycle advances ovulation and completion of the cycle by 1 day in the majority of animals (75 and 80%, respectively). Progesterone (250 μg) administered with APS eliminated the effect of the antiserum. Within 2 h after administration of APS, levels of both follicle–stimulating hormone (FSH) and luteinizing hormone (LH) elevated significantly, while significant elevation of plasma estradiol above the control value followed as late as 36 h after the treatment. None of the 5–day cyclic rats treated with APS showed ovulatory increases of FSH and LH at 1700 h on the second day of diestrus, although 3 of the 4 animals receiving the same treatment ovulated by 1100 h o following day. The onset of ovulatory release of gonadotropins might have delayed for several hours in these animals. These results indicate that recur of the 5–day cycle is due to an elevated progesterone secretion on the mc of diestrus, and suggest that a prolongation of luteal progesterone secreti an estrous cycle suppresses gonadotropin secretion. Rather than di blocking the estrogen triggering of ovulatory LH surge, the prolonged sec of luteal progesterone may delay the estrogen secretion itself, which decreas threshold of the neural and/or hypophyseal structures for ovulatory LH r

22438. IMBERT–TEBOUL, M.*, S. SIAUME and F. MOREL. Physiologie Cellulaire, College Fr., 11 Place Marcelin Berthelot, 75231 Cedex 05, Fr.) MOL CELL ENDOCR 45(1): 1–10. 1986. **Sites of prostag E_2 synthesis along the rabbit nephron.**—The purpose of this study v establish whether the nephron segments recognized as PGE_2 target sites rabbit, i.e., the proximal tubule, the thick ascending limb and the col tubule, are also sites of PGE_2 production. We therefore developed a mi munoassay sensitive enough to allow the measurement of PGE_2 on mi sected tubular segments about 1 mm in length. Under the conditions us min incubation at 20° C), a basal rate of PGE_2 production was measured cortical (CCT) and medullary portions of the collected tubule, as co expected. In the presence of 10^{-4} M sodium arachidonate, it was shown tl The thin descending limb (TDL) is also an active site of PGE_2 formation expressed per mm tubule length the amounts formed were lower in TD in CCT (14.1 ± 2.7 SE pg/mm, n = 5, vs 93.5 ± 10.7, n = 8). They wer comparable, however, when expressed per μg total proteins (0.70 ng in T 0.6 in CCT). (2) A slight PGE_2 production was noted in the connecting but it was likely due to contamination by adjacent CCT cells. (3) In th nephron segments, only negligible amounts of PGE_2 were formed, wh probably of no physiological significance.

22439. OTSUKI, MAKOTO*, YOSHINORI OKABAYASHI, AT OHKI, TORU OKA, MASATOSHI FUJII, TAKAHIKO NAKAI NOBUO SUGIURA, NOBORU YANAIHARA and SHIGEAKI (Second Dep. Internal Med., Kobe Univ. Sch. Med., Kobe 650, Japan. PHYSIOL 250(4 Part 1): G405–G411. 1986. **Action of cholecys analogues on exocrine and endocrine rat pancreas.**—In the present s have examined the abilities of cholecystokinin–(26–33)- [CCK–(26–33)–NH_2, CCK–8], nonsulfated CCK–(26–33)–NH_2 (de CCK–8), CCK–(30–33)–NH_2 (CCK–4), CCK–(26–33)–OH (dea CCK–8), and succinyl CCK–(27–31)–NH_2 (Suc–Des–Asp^6,Phe^7–CC stimulate exocrine pancreatic secretion from both isolated pancreatic a isolated perfused pancreas. We have also compared this action with the to cause insulin release. The modification of either the N– or C–termin acid residues of CCK–8 decreased in potency, but the magnitud stimulation of enzyme secretion caused by a maximally effective peptid tration was the same. The minimal effective concentration of CCK–8, d CCK–8, and CCK–4 for insulin release from the isolated rat pancre presence of 8.3 mM glucose was the same as that for pancreatic secretion. In contrast, the concentrations of deamidated CC Suc–Des–Asp^6–Phe^7–CCK–7 required to produce insulin release were 5 higher than those required to cause stimulation of pancreatic enzyme secretion. It is concluded therefore that the N–terminal 4–amino acid r the C–terminal 2–amino acid residues of CCK–8 are not essential for activity but do contribute to its potency. In addition, the C–terminal acid residues and an amide group in the C–terminal phenylalanine CCK–8 appear to be important determinants of the insulin–releasing the CCK peptides.

22440. KONTUREK, STANISLAW J.*, JANINA TASLI BILSKI, ALPHONS J. DE JONG, JAN B. M. J. JANSEN and C B. LAMERS. (Inst. Physiology, Academy Med., Krakow, Polan PHYSIOL 250(4 Part 1): G391–G397. 1986. **Physiological role and l of cholecystokinin release in dogs.**—In dogs with pancreatic fist feeding and intestinal perfusion with a sodium oleate or amino ac increased pancreatic protein secretion to ~ 110, 100, and 50%, resp the response to cholecystokinin (CCK) at a dose of 85 pmol · kg^{-1} · h CCK response increased in these studies to ~ 100, 180, and 40%, r of the value obtained with exogenous CCK, suggesting that, in additio other neurohormonal factors contribute to pancreatic enzyme s response to endogenous stimulants. Feeding and duodenal oleat or a also stimulate the release of pancreatic polypeptide (PP), which may in the control of pancreatic secretion in response to endogenous including CCK. Perfusion of the intact intestine with graded amoun (0.5–16 mmol/h) produced dose–dependent increments in plasma pancreatic protein similar to those obtained with intravenous infusio doses of CCK (0.85–255 pmol · kg^{-1} · h^{-1}). Oleate perfusion of isol loops (30 cm long) made of duodenejejunal (D–J) and ileal (I) se stimulated protein secretion but elevated plasma CCK only after perf D–J but not of the I loop. We conclude that 1) the endogenous C by various luminal stimulants drives the pancreatic protein secre release of CCK is confined to the foregut; and 3) PP concomitantly various intestinal stimulants may contribute to the control of pancre induced by endogenous CCK.